Amendments to the Specification:

Please amend page 10, the Table beginning on line 10 as follows:

| Inhibitor | Description/ | Mechanism of C1 |
|------------------------------|-------------------------------|--------------------------------|
| | comments | inhibition |
| C1 inhibitor | Plasma serine protease | Inhibits C1r and C1s activity |
| | inhibitor | |
| IVIg | Has broad activity | Blocks Clq ligand binding |
| CRT | Contains several active | May inhibit both Clq head |
| | domains | and C1q tail |
| C1Qr | Native C1q receptor | Binds Clq tail, inhibits Cl |
| | | formation |
| E. coli C1q | | Binds Clq tail, inhibits Cl |
| binding protein | | formation |
| gClqR | Native C1q receptor | Binds Clq head |
| Decorin | Matrix protein | Binds to Clq head and tail |
| | | preparations |
| Chondroitin sulphate | plasma proteoglycan/B cell- | Inhibits C1 formation |
| proteoglycan | secreted | |
| Surfactant protein A | Collectin present in | Inhibits Clq ligand binding |
| | the lung | and C1 formation |
| HNP-1 | Cytotoxic peptide produced by | Binds C1q tail and inhibits C1 |
| | neutrophils | formation |
| Peptide gC1q-R ₁₈ | Derived from gClqR | Not defined |
| (TDGDKAFVDFLSDEIKEE: | | |
| SEQ ID NO 1) | | |
| Peptide | Derived from CRT | Inhibits C1q ligand binding |
| KDIRCKDD (SEQ ID NO. 2) | | |
| Peptide | Derived from human IgG | Inhibits C1q ligand binding |
| AEAKAKA (SEQ ID NO. 3) | | |
| Peptide | Derived from human IgG1 | Not defined |
| VQVHNAKTKPR (SEQ ID | | |
| NO. 4) | | |

Table 1 (cont.)

| Inhibitor | Description/ comments | Mechanism of C1 inhibition |
|--|--------------------------|---|
| Peptide WY | Derived from human IgG | Inhibits C1q ligand binding |
| Peptide 2J (CEGPFGPRHDLTFCW SEQ ID NO. 5) | Synthetic peptide | Binds C1q head, inhibits ligand binding |
| ghB3 | Trimeric Clq B chain | Acts as a competitor for C1q binding |
| Peptide CBP2 LEQGENVFLQATLL (SEQ ID NO. 6) | Derived from Clq B chain | Acts as a competitor for C1q binding |

Please amend page 14, the paragraph beginning on line 12 and ending on page 15, line 6 as follows:

In this connection, peptides directly derived from IgG have been described to inhibit C1q, such as a 7-meric peptide (i.e. AEAKAKA SEQ ID NO. 3) containing the ExKxKx motif, an 11-meric peptide (VQVHNAKTKPR SEQ ID NO. 4) derived from IgG1 that is related to the same motif, and a dimeric peptide (WY, c.f Table 1). These peptides were able to inhibit activation of the classical complement pathway in several *in vitro* assays. However, the WY peptide also inhibits the alternative complement pathway.

Among 42 peptides selected from phage-displayed peptide libraries based on phage binding to human C1q, 20 peptides have been identified, which can inhibit the classical complement pathway in human serum. Remarkably, 13 out of these 20 peptides were able to inhibit the classical pathway as well as the alternative pathway in hemolytic assays, whereas 7 peptides specifically inhibited the classical pathway. Out of these peptides, the peptide 2J (CEGPFGPRHDLTFCW SEQ ID NO. 5) was selected. Peptide 2J is a strong inhibitor of C1q hemolytic function. Similar to the peptides with an IgG motif, peptide 2J binds to the globular head of C1q and inhibits the binding of C1q to IgG. In addition, peptide 2J inhibits C1q from human, primate and rodent origin.

Other selected peptides useful for inhibiting the classical pathway are CEGPFGPRHDLTFCW (SEQ ID NO. 5), CRWDGSWGEVRC (SEQ ID NO. 7), CMWVRMWGDVNC (SEQ ID NO. 8), CFWAGKFGLGTC (SEQ ID NO. 9), CKDRWVVEERCC (SEQ ID NO. 10), and CWNRFKKMDRC (SEQ ID NO. 11). Several other peptides can also be used, which act as a competitor for C1q binding and

are derived from the C1q B chain, e.g. the peptide CBP2 (LEQGENVFLQATLL $\underline{SEQ\ ID}\ \underline{NO.\ 6}$).